

CHAPTER 2

THE FUNDAMENTALS OF MENTAL HEALTH AND MENTAL ILLNESS

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CHAPTER 2

THE FUNDAMENTALS OF MENTAL HEALTH AND MENTAL ILLNESS

A vast body of research on mental health and, to an even greater extent, on mental illness constitutes the foundation of this Surgeon General's report. To understand and better appreciate the content of the chapters that follow, readers outside the mental health field may desire some background information. Thus, this chapter furnishes a "primer" on topics that the report addresses.

The chapter begins with an overview of research under way today that is focused on the neuroscience of mental health. Modern integrative neuroscience offers a means of linking research on broad "systems level" aspects of brain function with the remarkably detailed tools and findings of molecular biology. The report begins with a discussion of the brain because it is central to what makes us human and provides an understanding of mental health and mental illness. All of human behavior is mediated by the brain. Consider, for example, a memory that most people have from childhood—that of learning to ride a bicycle with the help of a parent or friend. The fear of falling, the anxiety of lack of control, the reassurances of a loved one, and the final liberating experience of mastery and a newly extended universe create an unforgettable combination. For some, the memories are not good ones: falling and being chased by dogs have left marks of anxiety and fear that may last a lifetime. Science is revealing how the skill learning, emotional overtones, and memories of such experiences are put together physically in the brain. The brain and mind are two sides of the same coin. Mind is not possible without the remarkable physical complexity that is built into the brain, but, in addition, the physical complexity of the

brain is useless without the sculpting that environment, experience, and thought itself provides. Thus the brain is now known to be physically shaped by contributions from our genes and our experience, working together. This strengthens the view that mental disorders are both caused and can be treated by biological and experiential processes, working together. This understanding has emerged from the breathtaking progress in modern neuroscience that has begun to integrate knowledge from biological and behavioral sciences.

An overview of mental illness follows the section on modern integrative brain science. The section highlights topics including symptoms, diagnosis, epidemiology (i.e., research having to do with the distribution and determinants of mental disorders in population groups, including various racial and ethnic minority groups), and cost, all of which are discussed in greater and more pointed detail in the chapters that follow. Etiology is the study of the origins and causes of disease, and that section reviews research that is seeking to define, with ever greater precision, the causes of mental disorders. As will be seen, etiology research examines fundamental biological, behavioral, and sociocultural processes, as well as a necessarily broad array of life events. The section on development of temperament reveals how mental health science has attempted over much of the past century to understand how biological, psychological, and sociocultural factors meld in health as well as in illness. The chapter then reviews research approaches to the prevention and treatment of mental disorders and provides an overview of mental health services and their delivery. Final sections cover the growing influence on the mental

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health field of the need for attention to cultural diversity, the importance of the consumer movement, and new optimism about recovery from mental illness—that is, the possibility of recovering one's life.

The Neuroscience of Mental Health¹

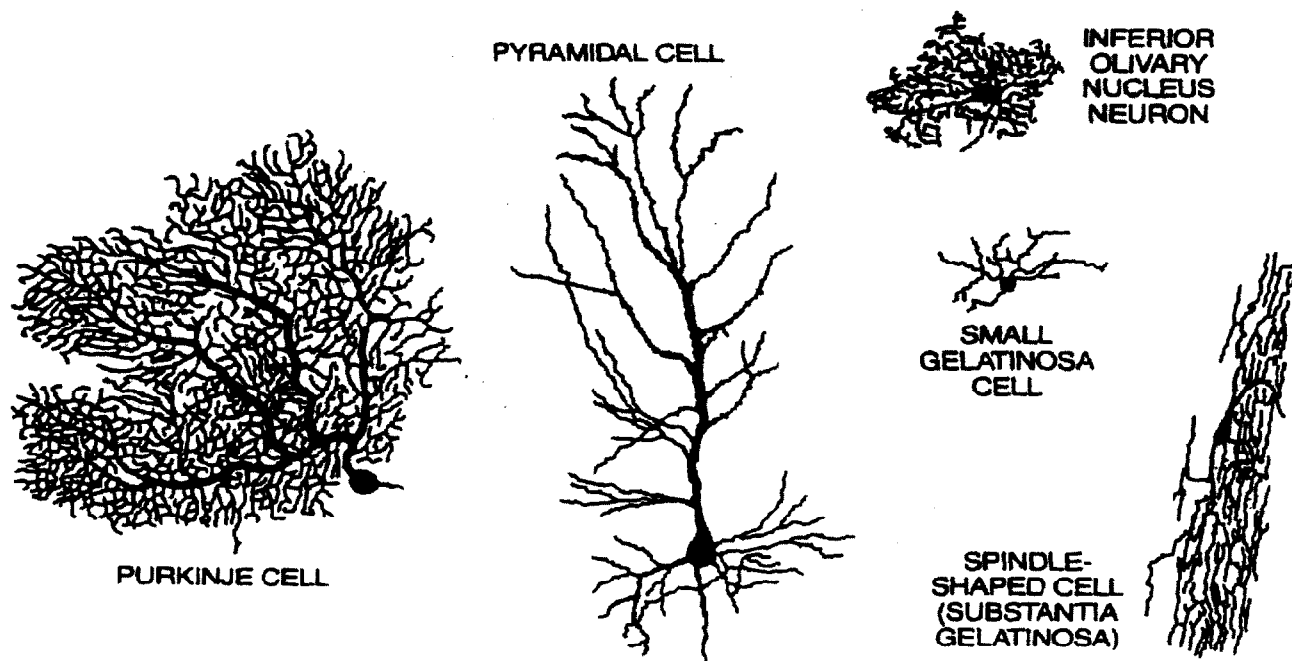
Complexity of the Brain I: Structural

As befits the organ of the mind, the human brain is the most complex structure ever investigated by our science. The brain contains approximately 100 billion nerve cells, or neurons, and many more supporting cells, or glia. In and of themselves, the number of cells

in this 3-pound organ reveal little of its complexity. Yet most organs in the body are composed of only a handful of cell types; the brain, in contrast, has literally thousands of different kinds of neurons, each distinct in terms of its chemistry, shape, and connections (Figure 2-1 depicts the structural variety of neurons). To illustrate, one careful, recent investigation of a kind of interneuron that is a small local circuit neuron in the retina, called the amacrine cell, found no less than 23 identifiable types.

But this is only the beginning of the brain's complexity.

Figure 2-1. Structural variety of neurons



Source: Fischbach, 1992, p. 53. (Permission granted: Patricia J. Wynne.)

¹ Special thanks to Steven E. Hyman, M.D., Director, National Institute of Mental Health, and Gerald D. Fischbach, M.D., Director, National Institute of Neurological Diseases and Stroke, for their contributions to this section.

The workings of the brain depend on the ability of nerve cells to communicate with each other. Communication occurs at small, specialized structures called synapses. The synapse typically has two parts. One is a specialized presynaptic structure on a terminal portion of the sending neuron that contains packets of signalling chemicals, or neurotransmitters. The second is a postsynaptic structure on the dendrites of the receiving neuron that has receptors for the neurotransmitter molecules.

The typical neuron has a cell body, which contains the genetic material, and much of the cell's energy-producing machinery. Emanating from the cell body are dendrites, branches that are the most important receptive surface of the cell for communication. The dendrites of neurons can assume a great many shapes and sizes, all relevant to the way in which incoming messages are processed. The output of neurons is carried along what is usually a single branch called the axon. It is down this part of the neuron that signals are transmitted out to the next neuron. At its end, the axon may branch into many terminals. (Figure 2-2.)

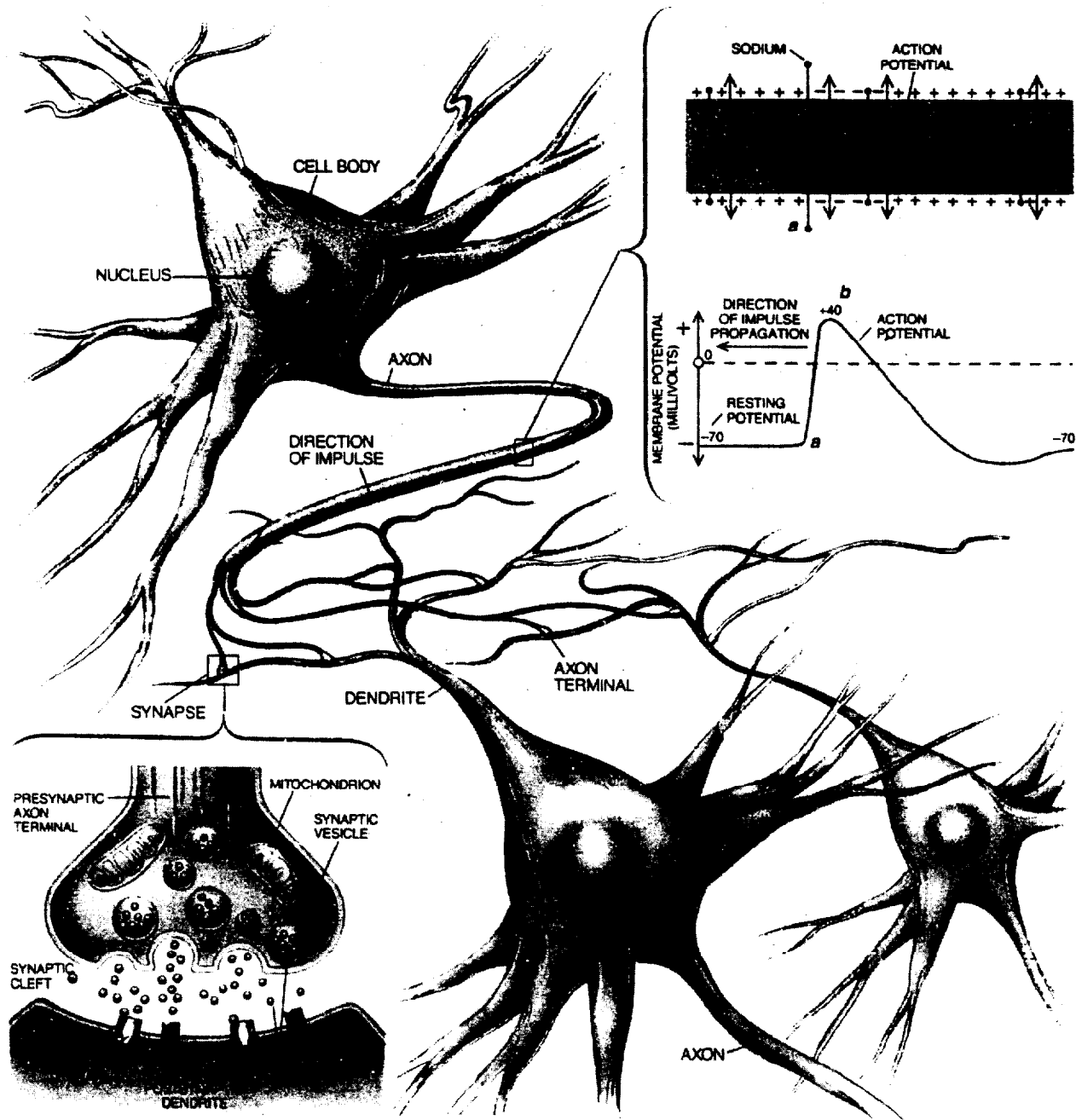
The usual form of communication involves electrical signals that travel within neurons, giving rise to chemical signals that diffuse, or cross, synapses, which in turn give rise to new electrical signals in the postsynaptic neuron. Each neuron, on average, makes more than 1,000 synaptic connections with other neurons. One type of cell—a Purkinje cell—may make between 100,000 and 200,000 connections with other neurons. In aggregate, there may be between 100 trillion and a quadrillion synapses in the brain. These synapses are far from random. Within each region of the brain, there is an exquisite architecture consisting of layers and other anatomic substructures in which synaptic connections are formed. Ultimately, the pattern of synaptic connections gives rise to what are called circuits in the brain. At the integrative level, large- and small-scale circuits are the substrates of behavior and of mental life. One of the most awe-inspiring mysteries of brain science is how neuronal activity within circuits gives rise to behavior and, even, consciousness.

The complexity of the brain is such that a single neuron may be part of more than one circuit. The organization of circuits in the brain reveals that the brain is a massively parallel, distributed information processor. For example, the circuits involved in vision receive information from the retina. After initial processing, these circuits analyze information into different streams, so that there is one stream of information describing what the visual object is, and another stream is concerned with where the object is in space. The information stream having to do with the identity of the object is actually broken down into several more refined parallel streams. One, for example, analyzes shape while another analyzes color. Ultimately, the visual world is resynthesized with information about the tactile world, and the auditory world, with information from memory, and with emotional coloration. The massively parallel design is a great pattern recognizer and very tolerant of failure in individual elements. This is why a brain of neurons is still a better and longer-lasting information processor than a computer.

The specific connectivity of circuits is, to some degree, stereotyped, or set in expected patterns within the brain, leading to the notion that certain places in the brain are specialized for certain functions (Figure 2-3). Thus, the cerebral cortex, the mantle of neurons with its enormous surface area increased by outpouchings, called gyri, and indentations, called sulci, can be functionally subdivided. The back portion of the cerebral cortex (i.e., the occipital lobe), for example, is involved in the initial stages of visual processing. Just behind the central sulcus is the part of the cerebral cortex involved in the processing of tactile information (i.e., parietal lobe). Just in front of the central sulcus is a part of the cerebral cortex involved in motor behavior (frontal lobe). In the front of the brain is a region called the prefrontal cortex, which is involved with some of the highest integrated functions of the human being, including the ability to plan and to integrate cognitive and emotional streams of information.

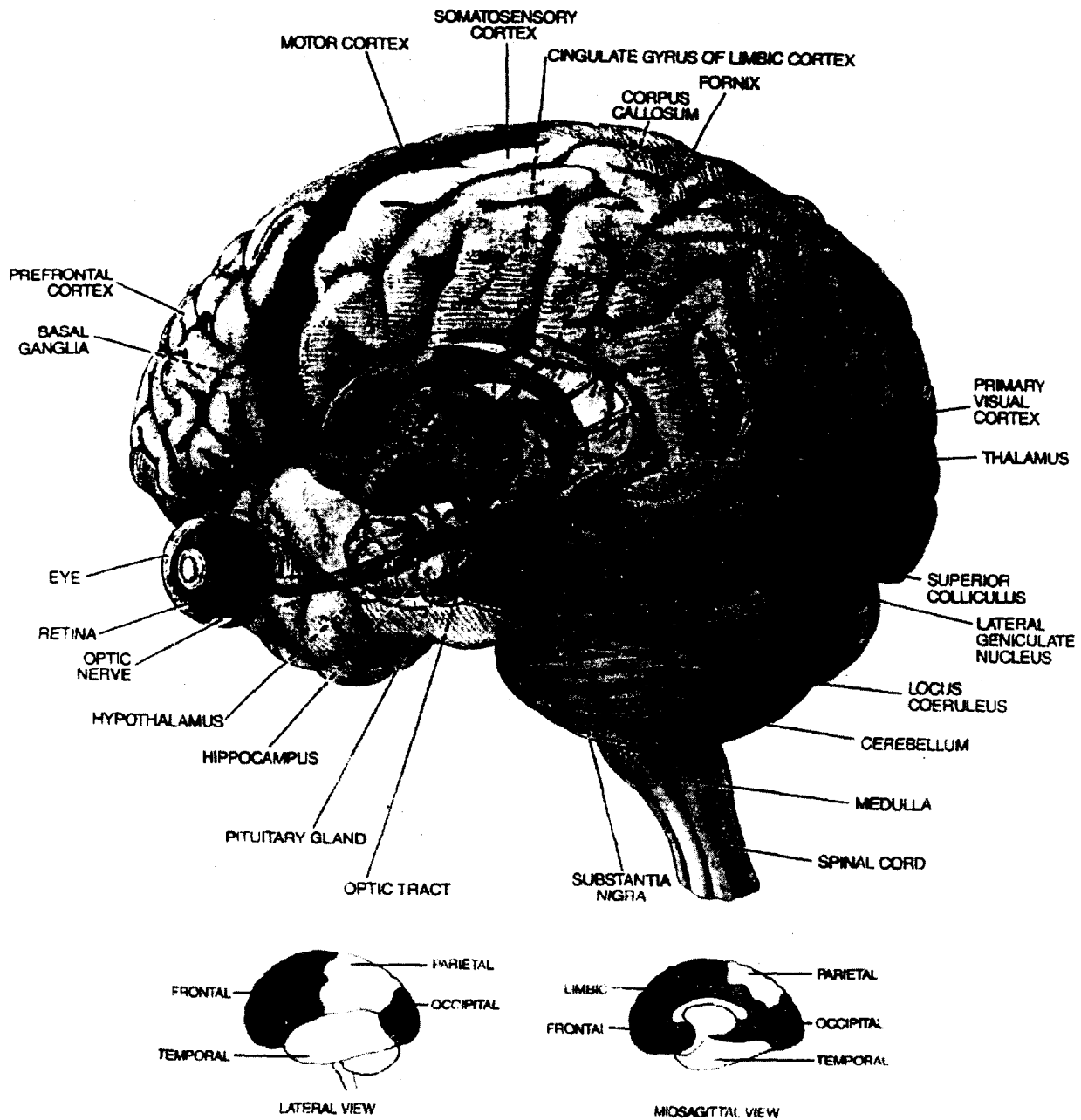
Beneath the cortex are enormous numbers of axons sheathed in the insulating substance, myelin. This sub-

Figure 2-2. How neurons communicate



Source: Fischbach, 1992, p. 52. (Permission granted: Tomo Narashima.)

Figure 2-3. The brain: Organ of the mind



Source: Fischbach, 1992, p. 51. (Permission granted: Carol Donner.)

cortical "white matter," so named because of its appearance on freshly cut brain sections, surrounds deep aggregations of neurons, or "gray matter," which, like the cortex, appears gray because of the presence of neuronal cell bodies. It is within this gray matter that

the brain processes information. The white matter is akin to wiring that conveys information from one region to another. Gray matter regions include the basal ganglia, the part of the brain that is involved in the initiation of motion and thus profoundly affected in

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Parkinson's disease, but that is also involved in the integration of motivational states and, thus, a substrate of addictive disorders. Other important gray matter structures in the brain include the amygdala and the hippocampus. The amygdala is involved in the assignment of emotional meaning to events and objects, and it appears to play a special role in aversive, or negative, emotions such as fear. The hippocampus includes, among its many functions, responsibility for initially encoding and consolidating explicit or episodic memories of persons, places, and things.

In summary, the organization of the brain at the cellular level involves many thousands of distinct kinds of neurons. At a higher integrative level, these neurons form circuits for information processing determined by their patterns of synaptic connections. The organization of these parallel distributed circuits results in the specialization of different geographic regions of the brain for different functions. It is important to state at this point, however, that, especially in younger individuals, damage to a particular brain region may yield adaptations that permit circuits spared the damage and, therefore, other regions of the brain, to pick up some of the functions that would otherwise have been lost.

Complexity of the Brain II: Neurochemical

Superimposed on this breathtaking structural complexity is the chemical complexity of the brain. As described above, electrical signals within neurons are converted at synapses into chemical signals which then elicit electrical signals on the other side of the synapse. These chemical signals are molecules called neurotransmitters. There are two major kinds of molecules that serve the function of neurotransmitters: small molecules, some quite well known, with names such as dopamine, serotonin, or norepinephrine, and larger molecules, which are essentially protein chains, called peptides. These include the endogenous opiates, Substance P, and corticotropin releasing factor (CRF), among others. All told, there appear to be more than 100 different neurotransmitters in the brain (Table 2-1 contains a selected list).

A neurotransmitter can elicit a biological effect in the postsynaptic neuron by binding to a protein called a neurotransmitter receptor. Its job is to pass the information contained in the neurotransmitter message from the synapse to the inside of the receiving cell. It appears that almost every known neurotransmitter has

Table 2-1. Selected neurotransmitters important in psychopharmacology

Excitatory amino acid

Glutamate

Inhibitory amino acids

Gamma aminobutyric acid

Glycine

Monoamines and related neurotransmitters

Norepinephrine

Dopamine

Serotonin

Histamine

Acetylcholine (quarternary amine)

Purine

Adenosine

Neuropeptides

Opioids

Enkephalins

Beta-endorphin

Dynorphin

Tachykinin

Substance P

Hypothalamic-releasing factors

Corticotropin-releasing hormone

more than one different kind of receptor that can confer rather different signals on the receiving neuron. Dopamine has 5 known neurotransmitter receptors; serotonin has at least 14.

Although there are many kinds of receptors with many different signaling functions, we can divide most neurotransmitter receptors into two general classes. One class of neurotransmitter receptor is called a ligand-gated channel, where "ligand" simply means a

molecule (i.e., a neurotransmitter) that binds to a receptor. When neurotransmitters interact with this kind of receptor, a pore within the receptor molecule itself is opened and positive or negative charges enter the cell. The entry of positive charge may activate additional ion channels that allow more positive charge to enter. At a certain threshold, this causes a cell to fire an action potential—an electrical event that leads ultimately to the release of neurotransmitter. By definition, therefore, receptors that admit positive charge are excitatory neurotransmitter receptors. The classic excitatory neurotransmitter receptors in the brain utilize the excitatory amino acids glutamate and, to a lesser degree, aspartate as neurotransmitters. Conversely, inhibitory neurotransmitters act by permitting negative charges into the cell, taking the cell farther away from firing. The classic inhibitory neurotransmitters in the brain are the amino acids gamma amino butyric acid, or GABA, and, to a lesser degree, glycine.

Most of the other neurotransmitters in the brain, such as dopamine, serotonin, and norepinephrine, and all of the many neuropeptides constitute the second major class. These are neither precisely excitatory nor inhibitory but rather act to produce complex biochemical changes in the receiving cell. Their receptors do not contain intrinsic ion pores but rather interact with signaling proteins, called “G proteins” found inside the cell membrane. These receptors thus are called G protein-linked receptors. The details are less important than understanding the general scheme. Stimulation of G protein-linked receptors alters the way in which receiving neurons can process subsequent signals from glutamate or GABA. To use a metaphor of a musical instrument, if glutamate, the excitatory neurotransmitter, is puffing wind into a flute or clarinet, it is the modulatory neurotransmitters such as dopamine or serotonin that might be seen as playing the keys and, thus, altering the melody via G protein-linked receptors.

The architecture of these systems drives home this point. The precise brain circuits that carry specific information about the world and that are involved in

precise point-to-point communication within the brain use excitatory or inhibitory neurotransmission. Examples of such circuits, which are massively parallel, can be found in the visual and auditory cortex. Overlying this pattern of precise, rapid (timing in the range of milliseconds) neurotransmission are the modulatory systems in the brain that use norepinephrine, serotonin, and dopamine. In each case, the neurotransmitter in question is made by a very small number of nerve cells clustered in a limited number of areas in the brain. Of the hundred billion neurons in the brain, only about 500,000, for example, make dopamine—that is, for every 200,000 cells in the brain, only one makes dopamine. Even fewer make norepinephrine. The cell bodies of the dopamine neurons are clustered in a few brain regions, most importantly, regions deep in the brain, in the *midbrain*, called the *substantia nigra*, and the *ventral tegmental area*. Norepinephrine neurons are made in the nucleus *locus coeruleus* even farther down in the brain stem in a structure called the *pons*. Serotonin is made by a somewhat larger number of nuclei but, still, not by many cells. Nuclei called the *raphe nuclei* spread along the brain stem. While each of these neurotransmitters is made by a small number of neurons with clustered cell bodies, each sends its axons branching throughout the brain, so that in each case a very small number of neurons, which largely appear to fire in unison when excited, influence almost the entire brain. This is not the picture of systems that are communicating precise bits of information about the world but rather are intrinsic modulatory systems that act via other G protein-linked receptors to alter the overall responsiveness of the brain. These neurotransmitters are responsible for brain states such as degree of arousal, ability to pay attention, and for putting emotional color or significance on top of cold cognitive information provided by precise glutaminergic circuits. It is no wonder that these modulatory neurotransmitters and their receptors are critical targets of medications used to treat mental disorders—for example, the antidepressant and antipsychotic drugs—and also are the targets of drugs of abuse.

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Complexity of the Brain III: Plasticity

The preceding paragraphs have illustrated the chemical and anatomic structure of the brain and, in so doing, provided some picture of its complexity as well as some picture of its function. The crowning complexity of the brain, however, is that it is not static. The brain is always changing. People learn so much and have so many distinct types of memory: conscious, episodic memory of the sort that is encoded initially in the hippocampus; memory of motor programs or procedures that are encoded in the striatum; emotional memories that can initiate physiologic and behaviorally adaptive repertoires encoded, for example, in the amygdala; and many other kinds. Every time a person learns something new, whether it is conscious or unconscious, that experience alters the structure of the brain. Thus, neurotransmission in itself not only contains current information but alters subsequent neurotransmission if it occurs with the right intensity and the right pattern. Experience that is salient enough to cause memory creates new synaptic connections, prunes away old ones, and strengthens or weakens existing ones. Similarly, experiences as diverse as stress, substance abuse, or disease can kill neurons, and current data suggest that new neurons continue to develop even in adult brains, where they help to incorporate new memories. The end result is that information is now routed over an altered circuit. Many of these changes are long-lived, even permanent. It is in this way that a person can look back 10 or 20 or 50 years and remember family, a home or school room, or friends. The general theme is that to really understand the kind of memory—indeed, any brain function—one must think at least at two levels: one, the level of molecular and cellular alterations that are responsible for remodeling synapses, and, two, the level of information content and behavior which circuits and synapses serve.

To summarize this section, scientists are truly beginning to learn about the structure and function of the brain. Its awe-inspiring complexity is fully consistent with the fact that it supports all behavior and mental life. Implied in the foregoing, is the fact that brains are built not only by genes—and again, it is the

lion's share of the 80,000 or so human genes that are involved in building a structure so complex as the brain. Genes are not by themselves the whole story. Brains are built and changed through life through the interaction of genes with environment, including experience. It is true that a set of genes might create repetitive multiples of one type of unit, yet the brain appears far more complex than that. It stands to reason that if 50,000 or 60,000 genes are involved in building a brain that may have 100 trillion or a quadrillion synapses, additional information is needed, and that information comes from the environment. It is this fundamental realization that is beginning to permit an understanding of how treatment of mental disorders works—whether in the form of a somatic intervention such as a medication, or a psychological “talk” therapy—by actually changing the brain.

Imaging the Brain

There are many exciting developments in brain science. Of great relevance to the study of mental function and mental illness is the ability to image the activity of the living human brain with technologies developed in recent decades, such as positron emission tomography scanning or functional magnetic resonance imaging. Such approaches can exploit surrogates of neuronal firing such as blood flow and blood oxygenation to provide maps of activity. As science learns more about brain circuitry and learns more from cognitive and affective neuroscience about how to activate and examine the function of particular brain circuits, differences between health and illness in the function of particular circuits certainly will become evident. We will be able to see the action of psychotropic drugs and, perhaps most exciting, we will be able to see the impact of that special kind of learning called psychotherapy, which works after all because it works on the brain.

Different brain chemicals, brain receptors, and brain structures will come up in the discussion of particular illnesses throughout this document. This section is meant to provide a panoramic, not a detailed, introduction and also to provide certain overarching lessons. When something is referred to as biological or brain-based, that is not shorthand for saying it is

genetic and, thus, predetermined; similarly, references to “psychological” or even “social” phenomena do not exclude biological processes. The brain is the great integrator, bringing together genes and environment. The study of the brain requires reducing problems initially to bite-sized bits that will allow investigators to learn something, but ultimately, the agenda of neuroscience is not reductionist; the goal is to understand behavior, not to put blinders on and try to explain it away. As the foregoing discussion illustrates, the brain also is complex. Thus, having a disease that affects one or even many critical circuits does not overthrow, except in extreme cases, such as advanced Alzheimer’s disease, all aspects of a person. Typically, people retain their personality and, in most cases, their ability to take responsibility for themselves.

In retrospect, early biological models of the mind seem impoverished and deterministic—for example, models that held that “levels” of a neurotransmitter such as serotonin in the brain were the principal influence on whether one was depressed or aggressive. Neuroscience is far beyond that now, working to integrate information coming “bottom-up” from genes and molecules and cells, with information flowing “top-down” from interactions with the environment and experience to the internal workings of the mind and its neuronal circuits. Ultimately, however, the goal is not only human self-understanding. In knowing eventually precisely what goes wrong in what circuits and what synapses and with what chemical signals, the hope is to develop treatments with greater effectiveness and with fewer side effects. Indeed, as the following chapters indicate, the hope is for cures and ultimately for prevention. There is every reason to hope that as our science progresses, we will achieve those goals.

Overview of Mental Illness

Mental illness is a term rooted in history that refers collectively to all of the diagnosable mental disorders. Mental disorders are characterized by abnormalities in cognition, emotion or mood, or the highest integrative aspects of behavior, such as social interactions or planning of future activities. These mental functions are all mediated by the brain. It is, in fact, a core tenet

of modern science that behavior and our subjective mental lives reflect the overall workings of the brain. Thus, symptoms related to behavior or our mental lives clearly reflect variations or abnormalities in brain function. On the more difficult side of the ledger are the terms disorder, disease, or illness. There can be no doubt that an individual with schizophrenia is seriously ill, but for other mental disorders such as depression or attention-deficit/hyperactivity disorder, the signs and symptoms exist on a continuum and there is no bright line separating health from illness, distress from disease. Moreover, the manifestations of mental disorders vary with age, gender, race, and culture. The thresholds of mental illness or disorder have, indeed, been set by convention, but the fact is that this gray zone is no different from any other area of medicine. Ten years ago a serum cholesterol of 200 was considered normal. Today, this same number alarms some physicians and may lead to treatment. Perhaps every adult in the United States has some atherosclerosis, but at what point does this move along a continuum from normal into the realm of illness? Ultimately, the dividing line has to do with severity of symptoms, duration, and functional impairment.

Despite the existence of a gray zone between health and illness, science can study the mechanisms by which illness occurs. Indeed, understanding mood regulation and its abnormalities, for example, proceeds independently from any set of diagnostic clinical criteria. Family studies, molecular genetics strategies, epidemiology, and the tools of clinical investigation tailored to specific populations are being used to investigate the mechanisms of mental illness. Specific manifestations of mental illness will be covered in succeeding pages.

This overview of mental illness focuses on those features of the disease process that are most common and characteristic of these disorders. The chapters that follow will present specific details about major categories of mental disorders that occur across the life span. The purpose here is to provide a framework upon which subsequent discussions of specific disorders can rest. The section leads with a descriptive overview of the cardinal manifestations, signs, and symptoms of

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mental disorders. It then describes how mental disorders are diagnosed and classified and provides an overview of the epidemiology and societal burden of mental disorders.

Manifestations of Mental Illness

Persons suffering from any of the severe mental disorders present with a variety of symptoms that may include inappropriate anxiety, disturbances of thought and perception, dysregulation of mood, and cognitive dysfunction. Many of these symptoms may be relatively specific to a particular diagnosis or cultural influence. For example, disturbances of thought and perception (psychosis) are most commonly associated with schizophrenia. Similarly, severe disturbances in expression of affect and regulation of mood are most commonly seen in depression and bipolar disorder. However, it is not uncommon to see psychotic symptoms in patients diagnosed with mood disorders or to see mood-related symptoms in patients diagnosed with schizophrenia. Symptoms associated with mood, anxiety, thought process, or cognition may occur in any patient at some point during his or her illness.

Anxiety

Anxiety is one of the most readily accessible and easily understood of the major symptoms of mental disorders. Each of us encounters anxiety in many forms throughout the course of our routine activities. It may often take the concrete form of intense fear experienced in response to an immediately threatening experience such as narrowly avoiding a traffic accident. Experiences like this are typically accompanied by strong emotional responses of fear and dread as well as physical signs of anxiety such as rapid heart beat and perspiration. Some of the more common signs and symptoms of anxiety are listed in Table 2-2. Anxiety is aroused most intensely by immediate threats to one's safety, but it also occurs commonly in response to dangers that are relatively remote or abstract. Intense anxiety may also result from situations that one can only vaguely imagine or anticipate.

Anxiety has evolved as a vitally important physiological response to dangerous situations that pre-

Table 2-2. Common signs of acute anxiety

- | |
|--|
| <ul style="list-style-type: none">• Feelings of fear or dread• Trembling, restlessness, and muscle tension• Rapid heart rate• Lightheadedness or dizziness• Perspiration• Cold hands/feet• Shortness of breath |
|--|

pares one to evade or confront a threat in the environment. The appropriate regulation of anxiety is critical to the survival of virtually every higher organism in every environment. However, the mechanisms that regulate anxiety may break down in a wide variety of circumstances, leading to excessive or inappropriate expression of anxiety. Specific examples include phobias, panic attacks, and generalized anxiety. In phobias, high-level anxiety is aroused by specific situations or objects that may range from concrete entities such as snakes, to complex circumstances such as social interactions or public speaking. Panic attacks are brief and very intense episodes of anxiety that often occur without a precipitating event or stimulus. Generalized anxiety represents a more diffuse and nonspecific kind of anxiety that is most often experienced as excessive worrying, restlessness, and tension occurring with a chronic and sustained pattern. In each case, an anxiety disorder may be said to exist if the anxiety experienced is disproportionate to the circumstance, is difficult for the individual to control, or interferes with normal functioning.

In addition to these common manifestations of anxiety, obsessive-compulsive disorder and post-traumatic stress disorder are generally believed to be related to the anxiety disorders. The specific clinical features of these disorders will be described more fully in the following chapters; however, their relationship to anxiety warrants mention in the present context. In the case of obsessive-compulsive disorder, individuals experience a high level of anxiety that drives their obsessional thinking or compulsive behaviors. When such an individual fails to carry out a repetitive

behavior such as hand washing or checking, there is an experience of severe anxiety. Thus while the outward manifestations of obsessive-compulsive disorder may seem to be related to other anxiety disorders, there appears to be a strong component of abnormal regulation of anxiety underlying this disorder. Post-traumatic stress disorder is produced by an intense and overwhelmingly fearful event that is often life-threatening in nature. The characteristic symptoms that result from such a traumatic event include the persistent reexperience of the event in dreams and memories, persistent avoidance of stimuli associated with the event, and increased arousal.

Psychosis

Disturbances of perception and thought process fall into a broad category of symptoms referred to as psychosis. The threshold for determining whether thought is impaired varies somewhat with the cultural context. Like anxiety, psychotic symptoms may occur in a wide variety of mental disorders. They are most characteristically associated with schizophrenia, but psychotic symptoms can also occur in severe mood disorders.

One of the most common groups of symptoms that result from disordered processing and interpretation of sensory information are the hallucinations. Hallucinations are said to occur when an individual experiences a sensory impression that has no basis in reality. This impression could involve any of the sensory modalities. Thus hallucinations may be auditory, olfactory, gustatory, kinesthetic, tactile, or visual. For example, auditory hallucinations frequently involve the impression that one is hearing a voice. In each case, the sensory impression is falsely experienced as real.

A more complex group of symptoms resulting from disordered interpretation of information consists of delusions. A delusion is a false belief that an individual holds despite evidence to the contrary. A common example is paranoia, in which a person has delusional beliefs that others are trying to harm him or her. Attempts to persuade the person that these beliefs are

unfounded typically fail and may even result in the further entrenchment of the beliefs.

Hallucinations and delusions are among the most commonly observed psychotic symptoms. A list of other symptoms seen in psychotic illnesses such as schizophrenia appears in Table 2-3. Symptoms of schizophrenia are divided into two broad classes: *positive* symptoms and *negative* symptoms. Positive symptoms generally involve the experience of something in consciousness that should not normally be present. For example, hallucinations and delusions represent perceptions or beliefs that should not normally be experienced. In addition to hallucinations and delusions, patients with psychotic disorders such as schizophrenia frequently have marked disturbances in the logical process of their thoughts. Specifically, psychotic thought processes are characteristically loose, disorganized, illogical, or bizarre. These disturbances in thought process frequently produce observable patterns of behavior that are also disorganized and bizarre. The severe disturbances of thought content and process that comprise the positive symptoms often are the most recognizable and striking features of psychotic disorders such as schizophrenia or manic depressive illness.

Table 2-3. Common manifestations of schizophrenia

Positive Symptoms	
•	Hallucinations
•	Delusions
•	Disorganized thoughts and behaviors
•	Loose or illogical thoughts
•	Agitation
Negative Symptoms	
•	Flat or blunted affect
•	Concrete thoughts
•	Anhedonia (inability to experience pleasure)
•	Poor motivation, spontaneity, and initiative

However, in addition to positive symptoms, patients with schizophrenia and other psychoses

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have been noted to exhibit major deficits in motivation and spontaneity that are referred to as negative symptoms. While positive symptoms represent the presence of something not normally experienced, negative symptoms reflect the absence of thoughts and behaviors that would otherwise be expected. Concreteness of thought represents impairment in the ability to think abstractly. Blunting of affect refers to a general reduction in the ability to express emotion. Motivational failure and inability to initiate activities represent a major source of long-term disability in schizophrenia. Anhedonia reflects a deficit in the ability to experience pleasure and to react appropriately to pleasurable situations. Positive symptoms such as hallucinations are responsible for much of the acute distress associated with schizophrenia, but negative symptoms appear to be responsible for much of the chronic and long-term disability associated with the disorder.

The psychotic symptoms represent manifestations of disturbances in the flow, processing, and interpretation of information in the central nervous system. They seem to share an underlying commonality of mechanism, insofar as they tend to respond as a group to specific pharmacological interventions. However, much remains to be learned about the brain mechanisms that lead to psychosis.

Disturbances of Mood

Most of us have an immediate and intuitive understanding of the notion of mood. We readily comprehend what it means to feel sad or happy. These concepts are nonetheless very difficult to formulate in a scientifically precise and quantifiable way; the challenge is greater given the cultural differences that are associated with the expression of mood. In turn, disorders that impact on the regulation of mood are relatively difficult to define and to approach in a quantitative manner. Nevertheless, dysregulation of mood and the expression of mood, or affect, represent a major category among mental disorders.

Disturbances of mood characteristically manifest themselves as a sustained feeling of sadness or sustained elevation of mood. As with anxiety and psychosis, disturbances of mood may occur in a variety of patterns associated with different mental disorders. The disorder most closely associated with persistent sadness is major depression, while that associated with sustained elevation or fluctuation of mood is bipolar disorder. The most common signs of these mood disorders are listed in Table 2-4. Along with the prevailing feelings of sadness or elation, disorders of mood are associated with a host of related symptoms that include disturbances in appetite, sleep patterns, energy level, concentration, and memory.

Table 2-4. Common signs of mood disorders

Symptoms Commonly Associated With Depression
• Persistent sadness or despair
• Insomnia (sometimes hypersomnia)
• Decreased appetite
• Psychomotor retardation
• Anhedonia (inability to experience pleasure)
• Irritability
• Apathy, poor motivation, social withdrawal
• Hopelessness
• Poor self-esteem, feelings of helplessness
• Suicidal ideation
Symptoms Commonly Associated With Mania
• Persistently elevated or euphoric mood
• Grandiosity (inappropriately high self-esteem)
• Psychomotor agitation
• Decreased sleep
• Racing thoughts and distractibility
• Poor judgment and impaired impulse control
• Rapid or pressured speech

It is not known why diverse functions such as sleep and appetite should be altered in disorders of mood. However, depression and mania are typically associated with characteristic changes in these basic functions. Mood appears to represent a complex group of behaviors and responses that undergo precise and tightly controlled regulation. Higher organisms that must adapt to changing environments depend on optimal control of basic functions such as sleep, appetite, sex, and physical activity. This regulation must adapt to diurnal and seasonal changes in the environment. In addition, more complex behaviors such as exploration, aggression, and social interaction must also undergo a similar, perhaps closely linked, regulation. In humans, these complex behaviors and their regulation are believed to be associated with the expression of mood. A depressed mood appears to reflect a kind of global damping of these functions, while a manic state may result from an excessive activation of these same functions. The mechanisms underlying the diverse changes associated with the mood disorders are largely unknown, but their appearance as clusters in specific disorders along with their collective response to specific therapeutics suggests a common mechanistic basis.

Disturbances of Cognition

Cognitive function refers to the general ability to organize, process, and recall information. Cognitive tasks may be subdivided into a large number of more specific functions depending on the nature of the information remembered and the circumstances of its recall. In addition, there are many functions commonly associated with cognition such as the ability to execute complex sequences of tasks. Disturbances of cognitive function may occur in a variety of disorders. Progressive deterioration of cognitive function is referred to as dementia. Dementia may be caused by a number of specific conditions including Alzheimer's disease (to be discussed in subsequent chapters). Impairment of cognitive function may also occur in other mental

disorders such as depression. It is not uncommon to find profound disturbances of cognition in patients suffering from severe mood disturbances. More recently, cognitive deficits have been reported in schizophrenia and now have become a major new topic of research. Lastly, cognitive impairment frequently occurs in a host of chemical, metabolic, and infectious diseases that exert an impact on the brain.

The manifestations of cognitive impairment can vary across an extremely wide range, depending on severity. Short-term memory is one of the earliest functions to be affected and, as severity increases, retrieval of more remote memories becomes more difficult. Attention, concentration, and higher intellectual functions can be impaired as the underlying disease process progresses. Language difficulties range from mild word-finding problems to complete inability to comprehend or use language. Functional impairments associated with cognitive deficits can markedly interfere with the ability to perform activities of daily living such as dressing and bathing.

Other Symptoms

Anxiety, psychosis, mood disturbances, and cognitive impairments are among the most common and disabling manifestations of mental disorders. It is important, however, to appreciate that mental disorders leave no aspect of human experience untouched. It is beyond the scope of the present chapter to detail the full spectrum of presentations of mental disorders. Other common manifestations include, for example, somatic or other physical symptoms and impairment of impulse control. Many of these issues will be touched upon in subsequent chapters with reference to specific disorders.

Diagnosis of Mental Illness

The foregoing discussion has suggested that the manifestations of mental disorders fall into a number of distinct categories such as anxiety, psychosis, mood disturbance, and cognitive

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deficits. These categories are broad, heterogeneous, and somewhat overlapping. Moreover, any particular patient may manifest symptoms from more than one of these categories. This is not unexpected, given the highly complex interactions that take place among the neurobiological and behavioral substrates that produce these symptoms. Despite these confounding difficulties, a systematic approach to the classification and diagnosis of mental illness has been developed. Diagnosis is essential in all areas of health for shaping treatment and supportive care, establishing a prognosis, and preventing related disability. Diagnosis also serves as shorthand to enhance communication, research, surveillance, and reimbursement.

The diagnosis of mental disorders is often believed to be more difficult than diagnosis of somatic, or general medical, disorders, since there is no definitive lesion, laboratory test, or abnormality in brain tissue that can identify the illness. The diagnosis of mental disorders must rest with the patients' reports of the intensity and duration of symptoms, signs from their mental status examination, and clinician observation of their behavior including functional impairment. These clues are grouped together by the clinician into recognizable patterns known as syndromes. When the syndrome meets all the criteria for a diagnosis, it constitutes a mental disorder. Most mental health conditions are referred to as disorders, rather than as diseases, because diagnosis rests on clinical criteria. The term "disease" generally is reserved for conditions with known pathology (detectable physical change). The term "disorder," on the other hand, is reserved for clusters of symptoms and signs associated with distress and disability (i.e., impairment of functioning), yet whose pathology and etiology are unknown.

The standard manual used for diagnosis of mental disorders in the United States is the *Diagnostic and Statistical Manual of Mental*

Disorders. Most recently revised in 1994, this manual now is in its fourth edition (American Psychiatric Association, 1994, hereinafter cited in this report as DSM-IV). The first edition was published in 1952 by the American Psychiatric Association; subsequent revisions, which were made on the basis of field trials, analysis of data sets, and systematic reviews of the research literature, have sought to gain greater objectivity, diagnostic precision, and reliability. DSM-IV organizes mental disorders into 16 major diagnostic classes listed in Table 2-5. For each disorder within a diagnostic class, DSM-IV enumerates specific criteria for making the diagnosis. DSM-IV also lists diagnostic "subtypes" for some disorders. A subtype is a subgroup within a diagnosis that confers greater specificity. DSM-IV is descriptive in its listing of symptoms and does not take a position about underlying causation.

Table 2-5. Major Diagnostic Classes of Mental Disorders (DSM-IV)

Disorders usually first diagnosed in infancy, childhood, or adolescence
Delerium, dementia, and amnestic and other cognitive disorders
Mental disorders due to a general medical condition
Substance-related disorders
Schizophrenia and other psychotic disorders
Mood disorders
Anxiety disorders
Somatoform disorders
Factitious disorders
Dissociative disorders
Sexual and gender identity disorders
Eating disorders
Sleep disorders
Impulse-control disorders
Adjustment disorders
Personality disorders

DSM-IV and its predecessors² represent a unique approach to diagnosis by a professional field. No other sphere of health care has created such an extensive compendium of all of its disorders with explicit diagnostic criteria. The World Health Organization's *International Classification of Diseases* (10th edition, 1992) is a valuable compendium of all diseases. Its mental health categories are expanded upon in DSM-IV. The *International Classification of Diseases* (ICD) is the official classification for mortality and morbidity statistics for all signatories to the U.N. Charter establishing the World Health Organization. ICD-9CM (9th edition, Clinical Modification, 1991) is still the official classification for the Health Care Financing Administration.

Knowledge about diagnosis continues to evolve. Evolution in the diagnosis of mental disorders generally reflects greater understanding of disorders as well as the influence of social norms. Years ago, for instance, addiction to tobacco was not viewed as a disorder, but today it falls under the category of "Substance-Related Disorders." Although DSM-IV strives to cover all populations, it is not without limitations. The difficulties encountered in diagnosing mental disorders in children, older persons, and racial and ethnic minority groups are discussed later in this chapter and throughout this report. Diagnosis rests on clinician judgment about whether clients' symptom patterns and impairments of functioning meet diagnostic criteria. Cultural differences in emotional expression and social behavior can be misinterpreted as "impaired" if clinicians are not sensitive to the cultural context and meaning of exhibited symptoms, a topic discussed later in this chapter in Overview of Cultural Diversity and Mental Health Services.

Epidemiology of Mental Illness

Few families in the United States are untouched by mental illness. Determining just how many people have mental illness is one of the many purposes of the field of epidemiology. Epidemiology is the study of patterns of disease in the population. Among the key terms of this discipline, encountered throughout this report, are *incidence*, which refers to new cases of a condition which occur during a specified period of time, and *prevalence*, which refers to cases (i.e., new and existing) of a condition observed at a point in time or during a period of time. According to current epidemiological estimates, at least one in five people has a diagnosable mental disorder during the course of a year (i.e., 1-year prevalence).

Epidemiological estimates have shifted over time because of changes in the definitions and diagnosis of mental health and mental illness. In the early 1950s, the rates of mental illness estimated by epidemiologists were far higher than those of today. One study, for example, found 81.5 percent of the population of Manhattan, New York, to have had signs and symptoms of mental distress (Srole, 1962). This led the authors of the study to conclude that mental illness was widespread. However, other studies began to find *lower* rates when they used more restrictive definitions that reflected more contemporary views about mental illness. Instead of classifying anyone with signs and symptoms as being mentally ill, this more recent line of epidemiological research only identified people as mentally ill if they had a *cluster* of signs and symptoms that, when taken together, impaired people's ability to function (Pasamanick, 1959; Weissman et al., 1978). By 1978, the President's Commission on Mental Health (1978) concluded conservatively that the annual prevalence of specific mental disorders in the United States was about 15 percent. This figure comports with recent estimates of the extent of mental illness in the population. Even as this figure has become more sharply delineated, the older and larger estimates underscore the magnitude of mental distress in the

² DSM-I (American Psychiatric Association, 1952), DSM-II (American Psychiatric Association, 1968), DSM-III (American Psychiatric Association, 1979), and DSM-III-R (American Psychiatric Association, 1987).

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population, which this report refers to as “mental health problems.”

Adults

The current prevalence estimate is that about 20 percent of the U.S. population are affected by mental disorders during a given year. This estimate comes from two epidemiologic surveys: the Epidemiologic Catchment Area (ECA) study of the early 1980s and the National Comorbidity Survey (NCS) of the early 1990s. Those surveys defined mental illness according to the prevailing editions of the *Diagnostic and Statistical Manual of Mental Disorders* (i.e., DSM-III and DSM-III-R). The surveys estimate that during a 1-year period, 22 to 23 percent of the U.S. adult population—or 44 million people—have diagnosable mental disorders, according to reliable, established criteria. In general, 19 percent of the adult U.S. population have a mental disorder alone (in 1 year); 3 percent have both mental and addictive disorders; and 6 percent have addictive disorders alone.³ Consequently, about 28 to 30 percent of the population have either a mental or addictive disorder (Regier et al., 1993b; Kessler et al., 1994). Table 2-6 summarizes the results synthesized from these two large national surveys.

Individuals with co-occurring disorders (about 3 percent of the population in 1 year) are more likely to experience a chronic course and to utilize services than are those with either type of disorder alone. Clinicians, program developers, and policy-makers need to be aware of these high rates of comorbidity—about 15 percent of those with a mental disorder in 1 year (Regier et al., 1993a; Kessler et al., 1996).

Based on data on functional impairment, it is estimated that 9 percent of all U.S. adults have the mental disorders listed in Table 2-6 and experience some significant functional impairment (National

Advisory Mental Health Council [NAMHC], 1993). Most (7 percent of adults) have disorders that persist for at least 1 year (Regier et al., 1993b; Regier et al., in press). A subpopulation of 5.4 percent of adults is considered to have a “serious” mental illness (SMI) (Kessler et al., 1996). Serious mental illness is a term defined by Federal regulations that generally applies to mental disorders that interfere with some area of social functioning. About half of those with SMI (or 2.6 percent of all adults) were identified as being even more seriously affected, that is, by having “severe and persistent” mental illness (SPMI) (NAMHC, 1993; Kessler et al., 1996). This category includes schizophrenia, bipolar disorder, other severe forms of depression, panic disorder, and obsessive-compulsive disorder. These disorders and the problems faced by these special populations with SMI and SPMI are described further in subsequent chapters. Among those most severely disabled are the approximately 0.5 percent of the population who receive disability benefits for mental health-related reasons from the Social Security Administration (NAMHC, 1993).

Children and Adolescents

The annual prevalence of mental disorders in children and adolescents is not as well documented as that for adults. About 20 percent of children are estimated to have mental disorders with at least mild functional impairment (see Table 2-7). Federal regulations also define a sub-population of children and adolescents with more severe functional limitations, known as “serious emotional disturbance” (SED).⁴ Children and adolescents with SED number approximately 5 to 9 percent of children ages 9 to 17 (Friedman et al., 1996b).

³ Although addictive disorders are included as mental disorders in the DSM classification system, the ECA and NCS distinguish between addictive disorders and (all other) mental disorders. Epidemiologic data in this report follow that convention.

⁴ The term “serious emotional disturbance” is used in a variety of Federal statutes in reference to children under the age of 18 with a diagnosable mental health problem that severely disrupts their ability to function socially, academically, and emotionally. The term does not signify any particular diagnosis; rather, it is a legal term that triggers a host of mandated services to meet the needs of these children.

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Table 2-6. Best estimate 1-year prevalence rates based on ECA and NCS, ages 18-54

	ECA Prevalence (%)	NCS Prevalence (%)	Best Estimate ** (%)
Any Anxiety Disorder	13.1	18.7	16.4
Simple Phobia	8.3	8.6	8.3
Social Phobia	2.0	7.4	2.0
Agoraphobia	4.9	3.7	4.9
GAD	(1.5)*	3.4	3.4
Panic Disorder	1.6	2.2	1.6
OCD	2.4	(0.9)*	2.4
PTSD	(1.9)*	3.6	3.6
Any Mood Disorder	7.1	11.1	7.1
MD Episode	6.5	10.1	6.5
Unipolar MD	5.3	8.9	5.3
Dysthymia	1.6	2.5	1.6
Bipolar I	1.1	1.3	1.1
Bipolar II	0.6	0.2	0.6
Schizophrenia	1.3	—	1.3
Nonaffective Psychosis	—	0.2	0.2
Somatization	0.2	—	0.2
ASP	2.1	—	2.1
Anorexia Nervosa	0.1	—	0.1
Severe Cognitive Impairment	1.2	—	1.2
Any Disorder	19.5	23.4	21.0

*Numbers in parentheses indicate the prevalence of the disorder without any comorbidity. These rates were calculated using the NCS data for GAD and PTSD, and the ECA data for OCD. The rates were not used in calculating the any anxiety disorder and any disorder totals for the ECA and NCS columns. The unduplicated GAD and PTSD rates were added to the best estimate total for any anxiety disorder (3.3%) and any disorder (1.5%).

**In developing best-estimate 1-year prevalence rates from the two studies, a conservative procedure was followed that had previously been used in an independent scientific analysis comparing these two data sets (Andrews, 1995). For any mood disorder and any anxiety disorder, the lower estimate of the two surveys was selected, which for these data was the ECA. The best estimate rates for the individual mood and anxiety disorders were then chosen from the ECA only, in order to maintain the relationships between the individual disorders. For other disorders that were not covered in both surveys, the available estimate was used.

Key to abbreviations: ECA, Epidemiologic Catchment Area; NCS, National Comorbidity Study; GAD, generalized anxiety disorder; OCD, obsessive-compulsive disorder; PTSD, post-traumatic stress disorder; MD, major depression; ASP, antisocial personality disorder.

Source: D. Regier, W. Narrow, & D. Rae, personal communication, 1999

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Table 2-7. Children and adolescents ages 9 to 17 with mental or addictive disorders,* combined MECA sample

	Prevalence (%)
Anxiety disorders	13.0
Mood disorders	6.2
Disruptive disorders	10.3
Substance use disorders	2.0
Any disorder	20.9

*Disorders include diagnosis-specific impairment and Child Global Assessment Scale ≤ 70 (mild global impairment).

Source: Shaffer et al., 1996

Not all mental disorders identified in childhood and adolescence persist into adulthood, even though the prevalence of mental disorders in children and adolescents is about the same as that for adults (i.e., about 20 percent of each age population). While some disorders do continue into adulthood, a substantial fraction of children and adolescents recover or “grow out of” a disorder, whereas, a substantial fraction of adults develops mental disorders in adulthood. In short, the nature and distribution of mental disorders in young people are somewhat different from those of adults.

Older Adults

The annual prevalence of mental disorders among older adults (ages 55 years and older) is also not as well documented as that for younger adults. Estimates generated from the ECA survey indicate that 19.8 percent of the older adult population have a diagnosable mental disorder during a 1-year period (Table 2-8). Almost 4 percent of older adults have SMI, and just under 1 percent has SPMI (Kessler et al., 1996); these figures do not include individuals with severe cognitive impairments such as Alzheimer’s disease.

Future Directions for Epidemiology

The epidemiology of mental disorders is somewhat handicapped by the difficulty of identifying a “case” of a mental disorder. “Case” is an

Table 2-8. Best estimate prevalence rates based on Epidemiologic Catchment Area, age 55+

	Prevalence (%)
Any Anxiety Disorder	11.4
Simple Phobia	7.3
Social Phobia	1.0
Agoraphobia	4.1
Panic Disorder	0.5
Obsessive-Compulsive Disorder	1.5
Any Mood Disorder	4.4
Major Depressive Episode	3.8
Unipolar Major Depression	3.7
Dysthymia	1.6
Bipolar I	0.2
Bipolar II	0.1
Schizophrenia	0.6
Somatization	0.3
Antisocial Personality Disorder	0.0
Anorexia Nervosa	0.0
Severe Cognitive Impairment	6.6
Any Disorder	19.8

Source: D. Regier, W. Narrow, & D. Rae, personal communication, 1999

epidemiological term for someone who meets the criteria for a disease or disorder. It is not always easy to establish a threshold for a mental disorder, particularly in light of how common symptoms of mental distress are and the lack of objective, physical symptoms. It is sometimes difficult to determine when a set of symptoms rises to the level of a mental disorder, a problem that affects other areas of health (e.g., criteria for certain pain syndromes). In many cases, symptoms are not of sufficient intensity or duration to meet the criteria for a disorder and the threshold may vary from culture to culture.

Diagnosis of mental disorders is made on the basis of a multidimensional assessment that takes into account observable signs and symptoms of

illness, the course and duration of illness, response to treatment, and degree of functional impairment. One problem has been that there is no clearly measurable *threshold* for functional impairments. Efforts are currently under way in the epidemiology of mental disorders to create a threshold, or agreed-upon minimum level of functional limitation, that should be required to establish a “case” (i.e., a clinically significant condition). Epidemiology reflecting the state of psychiatric nosology during the past two decades has focused primarily on symptom clusters and has not uniformly applied—or, at times, even measured—the level of dysfunction. Ongoing reanalyses of existing epidemiological data are expected to yield better understanding of the rates of mental disorder and dysfunction in the population.

Another limitation of contemporary mental health knowledge is the lack of standard measures of “need for treatment,” particularly those which are culturally appropriate. Such measures are at the heart of the public health approach to mental health. Current epidemiological estimates therefore cannot definitively identify those who are in need of treatment. Other estimates presented in Chapter 6 indicate that some individuals with mental disorders are in treatment and others are not; some are seen in primary care settings and others in specialty care. In the absence of valid measures of need, rates of disorder estimated in epidemiological surveys serve as an imperfect proxy for the need for care and treatment (Regier et al., in press).

Subsequent sections of this report reveal the population basis of our understanding of mental health. Where appropriate, the report discusses mental health and illness across the entire population. At other times, the focus is on care in specialized mental health settings, primary health care, schools, the criminal justice system, and even the streets. A mainstream public health and population-based perspective demands such a broad view of mental health and mental illness.

Costs of Mental Illness

The costs of mental illness are exceedingly high. Although the question of cost is discussed more fully in Chapter 6, a few of the central findings are presented here. The direct costs of mental health services in the United States in 1996 totaled \$69.0 billion. This figure represents 7.3 percent of total health spending. An additional \$17.7 billion was spent on Alzheimer’s disease and \$12.6 billion on substance abuse treatment. Direct costs correspond to spending for treatment and rehabilitation nationwide.

When economists calculate the costs of an illness, they also strive to identify indirect costs. Indirect costs can be defined in different ways, but here they refer to lost productivity at the workplace, school, and home due to premature death or disability. The indirect costs of mental illness were estimated in 1990 at \$78.6 billion (Rice & Miller, 1996). More than 80 percent of these costs stemmed from disability rather than death because mortality from mental disorders is relatively low.

Overview of Etiology

The precise causes (etiology) of most mental disorders are not known. But the key word in this statement is *precise*. The *precise* causes of most mental disorders—or, indeed, of mental health—may not be known, but the broad forces that shape them *are* known: these are biological, psychological, and social/cultural factors.

What is most important to reiterate is that the causes of health and disease are generally viewed as a product of the *interplay* or *interaction* between biological, psychological, and sociocultural factors. This is true for all health and illness, including mental health and mental illness. For instance, diabetes and schizophrenia alike are viewed as the result of interactions between biological, psychological, and sociocultural influences. With these disorders, a biological predisposition is necessary but not sufficient to explain their occurrence (Barondes, 1993). For other disorders,

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a psychological or sociocultural cause may be necessary, but again not sufficient.

As described in the section on modern neuroscience, the brain and behavior are inextricably linked by the plasticity of the nervous system. The brain is the organ of mental function; psychological phenomena have their origin in that complex organ. Psychological and sociocultural phenomena are represented in the brain through memories and learning, which involve structural changes in the neurons and neuronal circuits. Yet neuroscience does not intend to reduce all phenomena to neurotransmission or to reinterpret them in a new language of synapses, receptors, and circuits. Psychological and sociocultural events and phenomena continue to have meaning for mental health and mental illness.

Much of the research that is presented in the remainder of this report draws on theories and investigations that predate the more modern view of integrative neuroscience. It is still meaningful, however, to speak of the interaction of biological and psychological and sociocultural factors in health and illness. That is where the overview of etiology begins—with the biopsychosocial model of disease, followed by an explanation of important terms used in the study of etiology. Then, against the backdrop of the introductory section on brain and behavior, the following sections address biological and psychosocial influences on mental health and mental illness, a separation that reflects the distinctive research perspectives of past decades. The overview of etiology draws to a close with a discussion of the convergence of biological and psychosocial approaches in the study of mental health and mental illness.

Biopsychosocial Model of Disease

The modern view that many factors interact to produce disease may be attributed to the seminal work of George L. Engel, who in 1977 put forward the *Biopsychosocial Model of Disease* (Engel, 1977). Engel's model is a framework, rather than a set of detailed hypotheses, for understanding health

and disease. To many scientists, the model lacks sufficient specificity to make predictions about the given cause or causes of any one disorder. Scientists want to find out what *specifically* is the contribution of different factors (e.g., genes, parenting, culture, stressful events) and *how* they operate. But the purpose of the biopsychosocial model is to take a broad view, to assert that simply looking at biological factors alone—which had been the prevailing view of disease at the time Engel was writing—is not sufficient to explain health and illness.

According to Engel's model, biopsychosocial factors are involved in the causes, manifestation, course, and outcome of health and disease, including mental disorders. The model certainly fits with common experience. Few people with a condition such as heart disease or diabetes, for instance, would dispute the role of stress in aggravating their condition. Research bears this out and reveals many other relationships between stress and disease (Cohen & Herbert, 1996; Baum & Posluszny, 1999).

One single factor in isolation—biological, psychological, or social—may weigh heavily or hardly at all, depending on the behavioral trait or mental disorder. That is, the relative importance or role of any one factor in causation often varies. For example, a personality trait like extroversion is linked strongly to genetic factors, according to identical twin studies (Plomin et al., 1994). Similarly, schizophrenia is linked strongly to genetic factors, also according to twin studies (see Chapter 4). But this does not mean that genetic factors completely preordain or fix the nature of the disorder and that psychological and social factors are unimportant. These social factors modify expression and outcome of disorders. Likewise, some mental disorders, such as post-traumatic stress disorder (PTSD), are clearly caused by exposure to an extremely stressful event, such as rape, combat, natural disaster, or concentration camp (Yehuda, 1999). Yet not everyone develops PTSD after such exposure. On average, about 9